UCLA Health

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Abstract Form

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Project Title:	Ventricular Fibrillation in the Setting of Hypokalemic Periodic Paralysis

Research Category (please check one):

☐	**Original Research**	☒	**Clinical Vignette**	☐	**Quality Improvement**	☐	**Medical Education Innovation**

Abstract

Introduction:
Potassium derangements are a well-known cause of cardiac arrhythmias. Hypokalemia can lead to a variety of cardiac arrythmias such as sinus bradycardia, atrioventricular block, and ventricular fibrillation; it can additionally present with symptoms such as muscle weakness and dysfunction. Periodic paralyses are a broad category of neuromuscular disorders that cause painless muscle weakness, and hypokalemic periodic paralysis is one of the most common subtypes with an estimated prevalence of 1 in 100,000.

Case Report:
We present a case of a 32-year-old man with history significant for pemphigus vulgaris presenting with witnessed ventricular fibrillation after complaining of muscle weakness after a routine rituximab infusion treatment. Return of spontaneous circulation was achieved and the patient was intubated before admission to the MICU. His labs showed a severe metabolic acidosis with a hemolyzed potassium of 1.9, with the etiology of cardiac arrest being presumed due to his profound hypokalemia.

Upon gathering additional history, the patient apparently periodically experienced lower extremity muscle weakness but an etiology was never established. Given his electrolyte derangement on presentation, this raised concern for hypokalemic periodic paralysis. Additional laboratory studies revealed a TSH < 0.001 with a free T3 of 10.2 and T4 of 3.59. He was managed with aggressive potassium replacement (410 mEq total), propranolol, methimazole, and hydrocortisone to address both his hypokalemia as well as his hyperthyroidism. He ultimately returned to baseline and was discharged on hospital day ten with methimazole and propranolol as well as outpatient endocrinology follow up.

Discussion:
Hypokalemic periodic paralysis has been typically characterized by an autosomal dominant mutation. However, there have been reports of sporadic cases in the setting of thyrotoxicosis as well, likely due to high circulating levels of thyroid hormones that augment the Na^+K^+ATPase activity, ultimately shifting potassium into cells and causing transient hypokalemia. Attacks of myopathy are usually precipitated by stress, such as exercise, and are varied in their presentation: milder attacks can present with muscle weakness, while more severe attacks can affect the intercostal muscles and cause difficulty coughing. The diagnosis is made by checking a potassium level during an attack and is supported by having normal levels between attacks.

The mainstay of treatment during acute attacks is to replete potassium, however long-term management involves treating the underlying etiology to prevent future attacks. Our patient was able to return to baseline with potassium replacement while in the inpatient setting. This case illustrates the importance of potassium homeostasis in the body and the wide variety of effects that disrupting the homeostasis can have on the body. It is important in both the inpatient and outpatient settings to correct potassium abnormalities and any underlying causes, especially in the prevention of periodic paralysis attacks and life-threatening cardiac arrhythmias.